

## THE DETERMINANTS OF MORTALITY: CONFERENCE SUMMARY

Adriana Lleras-Muney  
July 26, 2004

The purpose of this conference was to assess alternative accounts of the determinants of mortality, which factors appear to explain mortality reductions over time as well as cross-sectional differences, both within and between countries; to attempt to resolve differences in the existing theories (by thinking about whether there might exist a unified framework to characterize mortality changes and differentials); and to identify areas for future research. A small group of distinguished participants represented a wide range of views and disciplinary perspectives.

The meeting was largely informal, and there was no presentation of papers. A number background papers were made available to participants in advance, and were frequently referred to in the discussion. No formal record was made, and the summary here touches all the issues without attempting to provide full accounts of what was actually said. It is organized by the names of the lead-off speakers for each session, but the text should not be taken as representing the views of the speakers.

### I. Samuel Preston

Since the mid 19<sup>th</sup> century, the world has experienced unprecedented increases in life expectancy. Throughout most of human history life expectancy remained constant at about 25 years. But between 1850 and 1900, it rose to 30 years and it reached 63 years by 2000. Mortality can be broadly thought of as a function of 4 major factors, socio-economic circumstances, public health environment, medical technologies and individual practices. The following reviews studies about the factors that contributed to the declines in mortality up to 1950.

Increases in life expectancy during this period were largely due to reductions in infant and childhood mortality. McKeown's work suggests that effective prophylaxis played little or no role in improving health up to 1950. He documented that the declines in mortality from infectious diseases (the major causes of mortality prior to 1950) preceded the introduction of vaccines or of medical treatments for these diseases. Most scholars today believe that public health and improved nutrition were responsible for the declines in mortality during this period.

Public health interventions, such as the provision of clean water, and the disposal of sewage, although preceding the germ theory of disease, became much more effective after it was understood and accepted. Cutler and Miller suggest that about ½ of the declines in mortality in the US from 1910-1930 were due to improved access to clean water. Fogel suggests that the implementation of clean water and sewage decreased inequality in infant mortality rates across cities in the US between 1900 and 1950.

Preston and Haines provides indirect evidence to show reductions in the mortality of the children of doctors immediately after the inception of the germ theory of disease. Evidence from developing countries tends to suggest that health campaigns against specific diseases, for example those to fight TB, smallpox, malaria in Sri Lanka, have at times made a large contribution to declines in mortality, though no one knows for sure how much of the mortality decline should be attributed to these “vertical” disease campaigns, to improvements in integrated health-delivery systems, or simply to increases in non-medical determinants, such as income and education, or the public health practices that income and education made possible.

The other major change during this period was improved nutrition. The work by Fogel documents that height and weight-for-height, both of which predict mortality, improved steadily in England and France during the 19<sup>th</sup> century. Although there have clearly been increases in height and weight-for-height, there is no direct evidence on the caloric consumption of individuals for these periods. It is also worth noting that diarrhea and infectious diseases in childhood impair the ability to turn food into nutrition, while poor nutrition often increases susceptibility to disease, so that it is always difficult to attribute credit to improved food supplies or better protection against disease, for example through public health.

There is also a strong relationship between income and life expectancy across countries over time and at a point in time. The Preston Curves (see Preston, 1975) show that as per capita income increases life expectancy increases as well, although the increases are much larger for initially poor countries. The estimates imply that increases in income are responsible for about 20% of the increases in life expectancy. This relationship does not however explain how income is translated into better health, although better nutrition, education, the sanitation projects are obvious possibilities. Also it is worth noting that the direction of causality is not known: health improvements are almost certainly increase income just as income improves health.

## Discussion

- Basic model: To the four major factors should be added endowment factors, such as genes. It should also include the stock of health since presumably a lot of the heterogeneity in health outcomes in a population at some point in time is due to the differences in their life experiences. Also we must allow for interaction between factors. For example, education may be effective because it helps spread new ways of doing things, such as good personal hygiene.
- Timing issues: To what extent are the causes of death determined by events that took place many years ago? To what extent do experiences *in utero* and in early childhood explain SES gradients in adulthood, the evidence in Case et al suggest that uterine environment affects health in adulthood but does not explain gradients? To what extent are the effects intergenerational? How do we distinguish period and cohort effects? It sometimes takes about 30 years for structure of society to absorb new innovations in knowledge such as the germ theory of disease.

- Anomalies: There are close to identical patterns in education gradients across different countries with widely different systems of medical care, should we conclude that medical care doesn't matter? Also why do we observe different patterns for men and women? Some of the differences are due to the fact that they have different causes of death. Women (and Blacks) have benefited more than men from maternity wards and accessing hospitals.

## II. Sir Michael Marmot

In spite of the large improvements in life expectancy we have witnessed, there remain large differences in health within societies that are related to the socio-economic status of individuals. Even more puzzling, there is some evidence that health gradients<sup>1</sup> (differences in health across SES groups within a country) have increased, even though access to health care and other material conditions have improved. For example in York in 1901 there were differences in the infant mortality of the servant keeping class compared to other classes but these differences were small compared to differences today.

How important is the role of income? Interestingly the effect of income on life expectancy is larger across individuals within countries, than across countries. In the US the mortality of individuals ages 45 to 64 earning 15,000 a year is twice as high as those earning 30,000 (PSID). There is no difference in life expectancy between countries with an income per capita of 15,000 or 30,000 per year. On the other hand it is not clear that we should spend more on health since there is no relationship between health expenditures and life expectancy across countries.

One possible explanation for these facts is that the role of income is different at low and high levels of income. Income below certain thresholds results in poor health because it curtails access to food, housing, sanitation and other goods. Absolute differences in income may explain most of the differences across countries and especially for poor countries.

Beyond a certain point, income per se may not improve one's access to health inputs. Rather it is a marker for one's position in society. Status may matter in determining health and thus may explain why we observe persistent differences across SES within developed countries. There is biological evidence consistent with the theory that rank affects health, through its effect on perceived control and stress. Animal experiments suggest that animals at the bottom of a hierarchy are less able to deal with stress and have higher mortality. The results from the Whitehall studies in England are consistent with these findings: individuals in low employment grades have markedly different levels of a variety of biological markers associated with stress and poor health (glucose, C reactive protein, interleukin 6, etc. Another illustration of the importance of stress is the

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<sup>1</sup> It is worth noting here that it matters whether one defines gradients as absolute or relative differences across groups. These measures often give different results but there is no consensus on which should be used.

experience of Eastern Europe and the Former Soviet Union where plausibly stress is associated with increases in cardiovascular disease.

The proposal is that in order to reduce disparities in health we should equalize chances in childhood; and improve conditions in the workplace, the structure of communities and the quality of life for older individuals.

#### Discussion

- Stress and SES: It is unclear what the cause and effect of these correlations are. Does stress response change as a result of time in a low SES position, or is it that poor stress responses results in low SES?
- Stress, SES and gender: does the theory explain differences between men and women? To some extent since there is evidence that stress affects males more than females.
- Not all the evidence is consistent with the status hypothesis. Life expectancy in the US and other western countries has increase in the last 30 years even though various likely stressors such as income inequality, crime and other measures of social capital have deteriorated.
- Aren't there still large differences in access to care within countries that could partially explain the relationship between SES and health? In England the evidence suggests that quantity of care that the poor receive is as great as the quantity of care that the rich receive, but it is much less clear whether this is true for quantity relative to needs, or for quality of care.

### III. Robert Fogel

Several anthropometric measures, height, weight-per-height and height-for-age, have been developed by medical and nutritional experts. These measures are related to the nutritional status of individuals and they have been shown to be related to mortality through a variety of mechanisms.

There is medical evidence suggesting that nutritional status is related to the body's ability to fight infectious diseases. Evidence from animal experiments suggests that iodine deficiency *in utero* (only) results in iodine deficiency and related health problems that are measurable up to three generations latter in rats.

Several studies by David Barker suggest that nutrition *in utero*, or lack thereof, can have consequences on the formation of organs related to chronic diseases developed late in life, such as cardiovascular disease. Consistent with Barker's work, the surviving children who were undernourished *in utero* during the Dutch famine had increased likelihood of coronary heart disease at age 50. Also, Doblhammer and Vaupel have shown a relationship between month at birth and longevity at age 50. The patterns the northern and southern hemispheres suggest that those born in the winter or spring months are more likely to die, presumably because the seasonal availability of food *in utero* affects adult mortality rates.

Finally it is worth noting that the effects of the Dutch famine on health were found not only on those exposed to the famines, but also on their children, although not until they themselves became parents, increasing the likelihood of premature fetal deaths and neonatal mortality.

Studies from Whitehall, studies that looked at slaves and population studies from several countries have found a robust relationship between birth weight, height and health. On average, low birth weight and stunting at maturity are associated with poor cognitive development and high blood pressure.

#### Discussion

- Nutrition and infectious disease. There is also some evidence that exposure to infectious diseases early in life can result in increased risk for chronic diseases in adulthood, as suggested by the work of Almond, who found that infants exposed to the influenza epidemic in 1918 had increased adult mortality rates. Infectious diseases, like diarrhea, affect the body's ability to metabolize food. So the relationship between infectious diseases and nutrition could be driven by the effect of infectious diseases on nutritional status.
- Dutch famine studies and influenza epidemic. Some of these findings are controversial. Studies of famines in Finland have not reproduced the results of the Dutch famine studies. The mechanisms for the intergenerational transmission in the Dutch famine studies are not known. It is also not clear whether it is nutrition *in utero* only that matters or whether other cohorts were also affected. The influenza epidemic is a difficult natural experiment because it followed immediately after the First World War, which had its own consequences for population health.
- Nutrition and SES. What is the effect of birth weight and malnutrition on educational attainment? Studies have found a strong relationship between nutritional status in uterus and cognition. In England birth weight has been correlated with lower test scores, especially for low SES parents. The reason apparently is that although all low birth weight children have lower cognition up to age two, high SES children catch up but low SES children do not. Case et al find however that low birth weight predicts health in adulthood and its effect is independent from that of education. Interestingly, uterine environment explains an increasing share of the variance in adult self reported health status as adults age, explaining about 9% by age 40, but it does not seem to reduce SES gradients in health.

#### IV. David Cutler

After 1950 life expectancy increases have been mostly due to the decline in mortality from chronic diseases among those 45 years old and above. Life expectancy has increased by about 8.8 years and about 5.2 years of the increase are due to declines in cardiovascular mortality. There have also been large reductions in neonatal mortality (deaths within 28 days of birth) related to innovations in neo-natal care. How much of the decline in mortality is related to medical technology? In particular how much is the

decline in cardiovascular (hereafter CVD) mortality due to new medication and treatments?

The Framingham studies showed that 1/3 of the decline in mortality was due to lower incidence of heart disease, 1/3 was the result of increased survival after a primary episode and 1/3 was due to increase survival thereafter. There have been two medical contributions in treating CVD: medications, especially those to lower blood pressure and intensive technologies to treat acute episodes such as bypass surgery. Using the results from the Framingham studies it is possible to break down the contribution of each of these factors, since we can use the results of the RCTs to estimate how they impact incidence and survival. Overall it appears that about 30% of the increase in life expectancy from reduced CVD is due to intense medical intervention (surgery, etc), 30% comes from medication, 10% comes from reduced smoking rates, and the rest are attributable to unobserved changes in behaviors. The same exercise can be repeated by looking at neonatal care.

The results from both cardiovascular and neonatal mortality analyses suggest that a large fraction of the increase in life expectancy since 1950, about 3.4 years out of an 8.8 increase, can be attributed to medical innovation.

#### Discussion

- Access and technology: it is not only technology that matters for mortality but who has access. To some extent it is an interaction between both, since it wouldn't be possible to increase life expectancy in the absence of technology to do so.
- Alternative explanations for the decrease in CVD: How much of the decline in CVD is due to increased diagnosis of milder heart attacks? The evidence from Framingham and other registries in the US suggest that the decline was real, not just a change in diagnostic technology. How much of the decline in heart disease is due to changes in risk factors? Aside from smoking, we don't know. For example we don't know if there have been any significant changes in the incidence of depression or diabetes.
- Technology and gradients: How much does technology explain cross section differences, for example in race? Not much. Changes in medical technology over time are relatively much larger than the changes in other health inputs, whereas in the cross section, the differences in the type of medical care across individuals are much smaller than the differences in other determinants of health. But wouldn't cross-sectional differences be explained because technology diffuses slowly (in time and space)?
- Technology and expenditures: If technology matters, why are health expenditures and life expectancy so poorly correlated across countries? One possible explanation is that quality of life, another critical dimension of health, is not measured by life expectancy. Another possibility is that at the margin, medical care makes only a small difference, even though it makes a large difference at the mean. Also there are some differences in how countries spend money in terms of allocating it towards treatment or prevention. Alternatively, there could be

inefficiencies in health care provision. In other words health expenditures do not reflect effectiveness of care.

Jonathan Skinner

Even though there seems to be agreement that medical innovation has been important in increasing life expectancy in the last 50 years, the role of access to health care is debated. There still exist large differences in access to care by SES. Many of these differences are differences in the *quality* of care individuals receive. Quality of care is largely determined by residential location.

Studying mortality from cardiovascular disease after hospitalization, Skinner et al find that blacks are more likely to go to hospitals with high mortality. And both blacks and whites are more likely to die when they are treated in predominantly black hospitals. So the evidence suggests not so much that individuals are treated differently according to race by the hospital, but more that they have access to hospitals of very different quality levels.

However, even though there are large differences in the quality of care that individuals obtain, explicit measures of quality can only account for a small fraction of the differences in health outcomes by race.

James Vaupel

Data for the 20th century shows that life expectancy in the “best practice country””, i.e. in the country with the largest life expectancy, has increased linearly, about 3 months per year or 2 ½ years per decade since 1840. Any model of mortality must account for this pattern. The data also show that countries have been converging towards the “best practice” life expectancy level.

There is other evidence of convergence in life expectancy. After German reunification the mortality rates of adults at all ages from east and west converged. This may suggest that innovation and information have diffused, reducing differences across countries.

Bruce Link

The theory of fundamental causes of disease explains why gradients move from one disease into another, and why they have existed for different diseases at different points in time. According to this theory, the particular mechanisms linking SES to disease are unimportant: over time those with resources or power will be able to use those resources to improve their health at a higher rate than those without resources. Smoking, exercise clean water, pollution are proximate causes. If we address these causes we will reduce inequities in the short run but in the long run, as new diseases emerge and new treatments are discovered, health inequities reappear. Resources are understood here to be “flexible” in that they can be used to provide an advantage in a variety of circumstances.

There is indeed some evidence that gradients emerge when new technologies become available. For example there were no differences in cardiovascular mortality between blacks and whites in 1950 but they are large differences now. There are other examples of diseases for which gradients emerged only after innovations in treatment are discovered, such as lung cancer and breast cancer. Smoking patterns also responded similarly after the Surgeon general report in 1964.

More generally, Link et al. show that gradients are larger for treatable than for untreatable diseases. This theory is different from the status and stress theory in that it does not emphasize stress or other psycho-social factors, which are proximate, not fundamental causes. It instead relies on the idea that individuals will always seek to advance themselves relative to others. According to fundamental cause theory gradients will reliably arise or become stronger when there are known and effective ways to avoid or treat the disease.

John Hobcraft

Work on developing countries has concentrated mostly on public interventions targeting infant health, immunizations and primary health care. However the importance of maternal education is well documented. There remain nonetheless many differences across countries that are not well understood. For example, why the elasticity of life expectancy with respect to income is so much larger for poor countries?

There have been a number of very successful countries, such as Costa Rica and China, that have achieved very large improvements in health at a very low cost. In general these success stories have occurred in countries with a strong political will to equalize access (and to improve education and nutrition). Perhaps it is not income that matters, but rather the structural organization of countries and their ability to mobilize resources to achieve particular goals. In that sense, perhaps what matters is the strength of government, institution and society.

Robert Wallace

There are many factors that have not been explored in thinking about the improvements in health in the last two centuries. The impact of clean water has been studied but the effects of clean air are not known. There have been changes in work conditions, jobs have become safer and they provide for insurance. “Dirty” jobs have been exported—they are now mostly in third world countries. The emergence of the energy economy has had wide impacts, including improvements in transportation and nutrition, as well as the availability of air-conditioning for the elderly. There has been an increase in the quality of products that are available, including improving the quality of the medical care. Practices related to land use and crowding have improved.

## DIRECTIONS FOR FUTURE WORK

### 1. Extending work beyond industrialized countries



The largest health gains are to be made in the developing world. Prior to the advent of AIDS, Africa's life expectancy had steadily converged towards that of developed countries—the precise causes of this convergence are still controversial. More generally, differences across countries are not well understood. For example, in Japan life expectancy has continued to increase in spite of increases in crime, and high smoking rates, cholesterol and low expenditures on health. Life expectancy in the US is 7 years lower than that of Japan and the gap seems to be widening, in spite of much larger health care expenditures per capita in the US. Costa Rica and Cuba have achieved large improvements in the health of their populations at very low costs. It would be important to understand what the systematic differences are. Hypothesis such as David Cutler's on the relative importance of behaviors and technology need to be taken to countries other than the US. There is likely to be a high payoff to all sorts of international comparisons of patterns of mortality. Disaggregation by sex is also likely to be informative, because behavioral patterns, such as smoking, differ by men and women, whereas medical technology differs much less. The convergence in life-expectancy and mortality rates across rich countries is not fully understood.

## 2. Understanding differences in behavior.

Both the status hypothesis and the fundamental-causes hypothesis suggest that it is necessary to further understand what drives differences across individuals with respect to health behaviors. Several important risks for disease are related to individual behaviors. For example, more educated individuals smoke at lower rates than less educated individuals, and they are more likely to wear their seat belts. They are also more likely to comply with medical treatments, or at least to be labeled as compliant by health providers. These behaviors are in turn reflected in their lower mortality rates. We do not understand the reasons why behavior differs and how to change this behavior. It is also important to note that gender and racial differences in health are large and not well-understood.

Interactions between factors, individual and social, have not been sufficiently explored. There were times in history where more affluent individuals could not improve their lot since there were no known methods to fight particular diseases. Conversely many individuals today die of preventable causes. Therefore it seems that the interaction between socioeconomic status and other factors must be important in determining gradients and their trends. But there is little evidence on interactions, except for the emerging evidence from genetics on the interaction between genes and the environment. The debate has focused mostly on separating and evaluating the impact of individual factors but this may not be possible or sensible.

## 3. Understanding the long term impacts of nutrition and other inputs early in life and across generations.

A potential consequence of the improved nutrition of adults and of the advances against infectious diseases in the early 20<sup>th</sup> century is that they may explain decreases in

mortality in latter periods, perhaps accounting for some of the increases in life expectancy since 1950. There is emerging evidence of intergenerational transmission of health. So improvements for one cohort may benefit subsequent cohorts. These lags are potentially long. It would be important to obtain more and better information about the events that may have affected individuals in utero and in their childhood, to relate it to the onset of disease in adulthood.

#### 4. Integrating health into our national health accounts.


GDP measures the contribution of better health to living standards by looking at changes in inputs in health, rather than outputs, such as increases in life expectancy. It is important to at least put bounds on the contribution of health care to GDP. Although there are many known difficulties in adding health gains into our national accounts, the suggestion is that integrating estimates of the improvements *disease by disease* would be valuable. For overall life-expectancy, we do not know how much is due to medical care, so that to attribute all of it to the healthcare sector almost certainly overstates its contribution to (extended) GDP. A disease by disease approach is likely to be less controversial, and will presumably identify areas where healthcare has negative value added.

#### 5-Reconciling trends and cross-section results

The factors that explain changes in mortality over time and those that explain differences within a population at a point in time appear different. One potential explanation is that the cross-sectional variation in factors that explain trends in small (relative to the time series variation) and vice-versa. For example, medical technology has changed a lot overtime but access to it may not be that different across regions or individuals at a given point in time. It should be possible to test this hypothesis. More generally, there may be alternative explanations for why the determinants of mortality appear to be different in the time series and in cross sections. It is important to understand why.

#### Conclusions

The conference was extremely successful in its format. The participants agreed to continue a conversation and find ways to build upon what was learned at the conference by sharing information within the group and making the findings of the conference available to a wider audience.



**THE DETERMINANTS OF MORTALITY**

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○ Agenda ○

**The Determinants of Mortality**

**Center for Health & Wellbeing**  
**300 Wallace Hall**  
**Princeton University**

**July 15 - 16th, 2004**

**Thursday, July 15**

6:00 pm Opening Dinner, Maclean House

**Friday, July 16**

8:30 am Coffee and continental breakfast

9:00 am Robert Fogel: *Nutrition*

9:30 am Discussion

10:00 am Michael Marmot: *Status*

10:30 am Break

10:45 am Discussion

11:15 am Sam Preston: *Public Health and risk factors*

11:45 am Discussion

12:15 pm Lunch

1:30 pm David Cutler: *Medical technology*

2:00 pm Discussion

2:30 pm Other accounts and discussion

*Shorter statements and discussion around other issues including:*

*Health in poor countries now (Hobcraft, Singer)*  
*Racial and geographical disparities (Skinner)*  
*Fundamental causes (Link)*  
*Public health and clinical advances (Wallace)*  
*Mortality changes among the elderly (Vaupel)*

4:30-5:00 pm

Summary and future plans

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